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EDITORIAL

Enigma of the Gastroesophageal Junction

FEW PARTS OF THE HUMAN BODY appear so innocuous and yet are so frustrating to anatomists, physiologists and clinicians as the gastroesophageal junction. Removed from its usual location and tacked out on a board, it is an unimpressive bit of tissue consisting of a tube merging into a pouch. The only other notable feature, visible on its inner side, is a rather abrupt change in the lining, which is pale and smooth at the upper, and darker and corrugated at the lower end. This change marks where squamous esophageal mucosa above meets glandular gastric mucosa below. In spite of its deceptively plain looks, the entire gastroesophageal junctional zone may be responsible, wholly or in part, for the major non-malignant disorders that affect man's esophagus: alkali-demanding heartburn, peptic esophagitis (which may hurt, bleed or stenose), lower esophageal (Schatzki) ring, and achalasia.

The very simplicity of the junctional zone defies an unexceptionable explanation of its contributions to the mechanisms of health and disease. There is no universally agreed-on benchmark to serve as a point of departure for describing the anatomy of the area. Although the word "cardia" is often used to indicate where esophagus opens into stomach, no serosal or mural structure exists to identify the cardia. Neither the shape nor the character of smooth muscle presents a definite line where the wall changes from tube to pouch. Under these circumstances, many rely on the gastroesophageal mucosal junction to separate the two organs. This practice serves convenience but does not necessarily enjoy authenticity. In man, many are convinced, the mucosal junction is not at the approximate location of the cardia, but well above it in a part of the junctional segment that is clearly tubular. In some animals, such as the horse and the rat, on the other hand, the mucosal junction is well down within the gastric pouch, and those who insist that this junction provides the discriminating landmark perforce apply the label esophagus to what is undeniably a sac-like upper half of stomach.

This anatomic no-man's land between stomach and gullet presents the author who would discuss esophageal disease with a major dilemma. Necessarily, if his discussion is to be anchored on basic anatomic and physiologic characteristics, he must draw a bold line and say, "On this side is esophagus; on that, stomach." Drawing a bold line across disputed territory, however, is hazardous. Zboralske and Friedland draw it as shown in Figure 1 of their well-balanced review of esophageal disorders in this issue (page 34). This line (labeled 4 in the figure) is identified as representing the "transverse mucosal fold," which, the authors say, demarcates the gastroesophageal junction and is known by a variety of synonyms, including "lower esophageal ring" or "cardia." Their text does not state definitely that this trans-

verse fold is created by the junction of gastric and esophageal mucosa, but the German words in Figure 3 identify it as such. That any mucosal fold, whatever its nature, exists normally and with any consistency so far on the gastric side of the human gastroesophageal junctional zone is doubtful. If a fold at the location shown in Figures 1 and 3 were as common and clear-cut as the authors suggest, would its illustration require a picture of a foreign anatomy specimen published in 1933?

Is this nit-picking? In its specific aspects, yes, but in its general implications, certainly no. For if an author faces a hazardous dilemma in drawing the exact location of the cardia, so does the clinician who seeks to interpret and treat esophageal symptoms rationally on the basis of abnormal gastroesophageal junctional mechanisms. In the absence of convincingly described and generally accepted anatomic features, the criteria used for diagnosing the presence of a sliding diaphragmatic hernia will be obligingly elastic; the radiologic demonstration of the lower esophageal ring above the level of the diaphragmatic hiatus will not be accepted by all as an unequivocal sign of diaphragmatic hernia; and it is disputable whether a gastroesophageal mucosal junction, found high up in the esophagus of a patient with chronic esophagitis (Barrett's esophagus), is there because of a congenital anomaly or because of upward migration resulting from repeated bouts of esophagitis.

Also shown in Zboralske and Friedland's diagram is an "inferior esophageal sphincter" (number 2) as well as a "vestibule" or "lower esophageal sphincter" (number 3)—which is shown as widely gaping! If all this is confusing, the reason is again anatomic: no clear-cut muscle fibers that might define a sphincter can be identified in the human gastroesophageal junction. Nevertheless, study of the distal esophagus *in situ* (principally by the technique of intralumenal manometry), and of muscle strips *in vitro,* show that the distal few centimeters of the tube portion in the junctional area are more irritable than the remaining esophageal muscle, respond differently, qualitatively and quantitatively, to pharmacologic stimuli, and tend to keep the distal esophageal lumen closed except when material passes normally from esophagus to stomach, or abnormally in a reverse direction. Non-relaxation of this sphincteric segment is held responsible for the distal esophageal obstruction in achalasia, whereas excessive relaxation, i.e., incompetence, is blamed in whole or in part for excessive gastroesophageal reflux. Hence, if treatment of these disorders is to improve, further studies will have to define, for the internist, the pharmacologic characteristics of the sphincter zone, and, for the surgeon, the influence exerted on sphincteric function by such paraesophageal structures as the phrenoesophageal membrane and the diaphragmatic hiatus. Observations in experimental animals should help to discover the needed knowledge, but in the most commonly used species striated esophageal muscle extends to, or nearly to, the gastroesophageal junction. Man's esophagus, in contrast, is almost unique among mammals in that only its upper third consists of striated muscle; the remainder and the entire gastroesophageal junction are made up of smooth muscle. The only reasonably available experimental animal with human-like esophagus, it is claimed, is the opossum!

Although no dramatic revelation of how the esophagus works has been forthcoming, the general clinical field of esophageal disease has been greatly clarified in the past 25 years by meticulous empirical observation. The varieties of diseases have been classified and hence are more readily recognized. Symptom patterns are now well enough defined to permit reasonably accurate diagnostic impressions, and the radiologic techniques so well illustrated by Zboralske and Friedland usually lead to definitive diagnoses. If doubt remains, endoscopy, biopsy, exfoliative cytology, esophageal sensitivity to acid perfusion (Bernstein test), and measurement of intraluminal pressure and acidity may be called upon. A patient who has difficult or painful swallowing should no longer mystify the doctor—even if that nondescript area where esophagus and stomach join remains an enigma.

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